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STATE-OF-THE-ART REVIEW

The Role of the Clinical Examination in Patients With Heart Failure

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CME/MOC Objectives for This Article: Upon completion of this activity, the learner should be able to: 1) describe key physical examination findings to separately define filling pressures and adequacy of perfusion in patients with heart failure; 2) identify high- and low-yield physical examination findings in heart failure that can practically be tested in the ambulatory or hospital setting; and 3) discuss potential scenarios in which "classic" physical examination findings in heart failure may be present in other clinical entities.

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The Role of the Clinical Examination in Patients With Heart Failure

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ABSTRACT

Despite advances in biomarkers and technology, the clinical examination (i.e., a history and physical examination) remains central in the management of patients with heart failure. Specifically, the clinical examination allows noninvasive assessment of the patient's underlying hemodynamic state, based on whether the patient has elevated ventricular filling pressures and/or an inadequate cardiac index. Such assessments provide important prognostic information and help guide therapeutic decision-making. Herein, the authors critically assess the utility of the clinical examination for these purposes and provide practical tips we have gleaned from our practice in the field of advanced heart failure. The authors note that the ability to assess for congestion is superior to that for inadequate perfusion. Furthermore, in current practice, elevated left ventricular filling pressures are inferred by findings related to an elevated right atrial pressure. They discuss an emerging classification system from the clinical examination that categorizes patients based on whether elevation of ventricular filling pressures occurs on the right side, left side, or both sides. (J Am Coll Cardiol HF 2018;6:543–51)

Despite technological advances, the clinical examination (i.e., a history and physical examination [H&P]) remains fundamental in the management of patients with heart failure (HF). As with any diagnostic test, the strengths and limitations of the clinical examination should be assessed critically. The purpose of this review is to summarize the current role of the clinical examination in determining the hemodynamic state and prognosis of patients with HF. Rather than providing a review of the comprehensive clinical examination, we focus on those components we believe are most useful in the routine care of such patients. We also highlight an emerging classification based on the pattern of ventricular filling pressure elevation.

The clinical examination can be used to assess the underlying hemodynamic state of patients with HF. Patients can be categorized based on clinician-estimated volume status (wet/dry) and perfusion status (warm/cold), as suggested by Dr. Lynne Stevenson (**Figure 1A**) (1). A patient is said to be wet if the estimated pulmonary capillary wedge pressure (PCWP) is ≥22 mm Hg; otherwise, the patient is considered dry. Likewise, if the estimated cardiac index (CI) is ≤2.2 l/min/m^2, a patient is said to be cold; otherwise, the patient is classified as warm. Thus, it becomes important to understand how each of these axes (volume status and perfusion) can be assessed by the clinical examination.

VOLUME STATUS

The determination of wet is based on the presence of any sign or symptom that is associated with elevated ventricular filling pressures. Such findings on the clinical examination include jugular venous distention (JVD), hepatojugular reflux (HJR), orthopnea, bendopnea, and a square-wave response in blood pressure (BP) during the Valsalva maneuver. The detection of volume depletion, the opposite side of the spectrum of volume status, can be assessed by orthostatic changes in BP and heart rate, which has been reviewed elsewhere (2).

JUGULAR VENOUS DISTENTION. JVD, or an elevated jugular venous pressure (JVP), has been found to be the most useful H&P finding for assessing ventricular filling pressures (3). JVD is described as an estimated JVP ≥10 cm H_2O (4). Estimates of cm H_2O can be converted to mm Hg using the relationship 1.36 cm H_2O = 1 mm Hg (5). If the internal jugular vein is difficult to appreciate, then assessment via the external jugular vein is acceptable (6). Some practical tips for the examination of the JVP are listed in **Table 1**.

JVD is common in patients admitted with decompensated HF (7,8). Assessment of the JVP can be used to estimate right atrial pressure (RAP) and guide therapy (6,7,9). Cardiology faculty were more accurate than fellows in estimating the JVP (10), thus emphasizing the importance of developing clinical examination skills. In ESCAPE (Evaluation Study

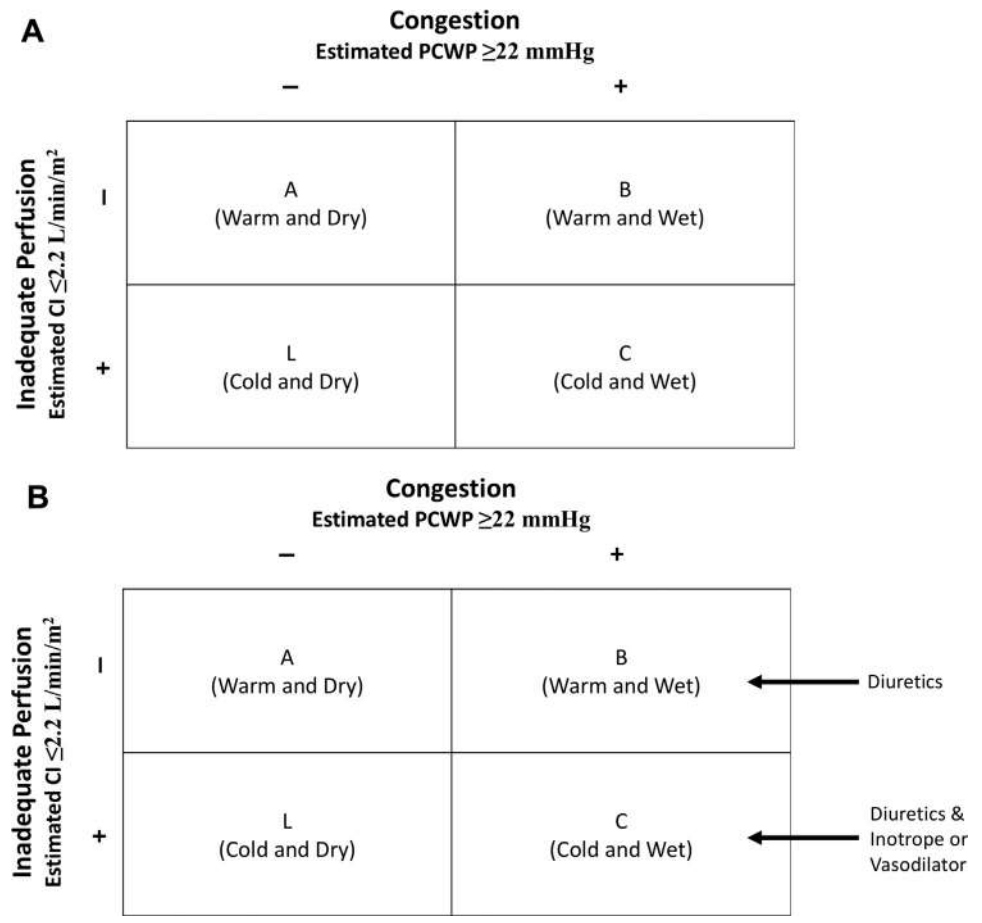
of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness), a pre-specified secondary objective was to assess the utility of skilled clinician assessment, via the H&P, of the hemodynamic status of patients with advanced HF (**Table 2**) (11). The JVP was categorized into 3 groups (<8, 8 to 12, >12 mm Hg), and these H&P-guided estimates were associated with the invasively measured RAP (area under the curve: 0.74) (7). Finally, in ESCAPE, JVD was 1 of only 2 parameters from the clinical examination (the other being orthopnea ≥2 pillows) that was associated with an elevated PCWP (7).

The prognostic implications of JVD have been examined. In SOLVD (Study of Left Ventricular Dysfunction), symptomatic patients with HF with JVD, an S_3 gallop (third heart sound), or both, were at significantly increased risk of hospitalization for HF, composite of death or hospitalization for HF, and death from pump failure (12). In patients with asymptomatic left ventricular dysfunction, both an S_3 gallop and JVD were associated with an increased risk of development of HF and the composite endpoint of death or development of HF in multivariable models (13). Subsequently, a propensity-matched population of patients with and without JVD did not demonstrate an association of JVD with outcomes (14). However, in a more recent study of more than 2,000 patients admitted with acute HF, JVD was associated with an increased risk of in-hospital adverse events and increased 30-day, 1-year, and 10-year all-cause mortality (4), findings consistent with those seen in SOLVD (12). An inspiratory increase in the venous pressure, equivalent to Kussmaul's sign, was also an adverse prognostic marker in

ABBREVIATIONS AND ACRONYMS

BP = blood pressure
CI = cardiac index
H&P = history and physical examination
HF = heart failure
HJR = hepatojugular reflux
JVD = jugular venous distention
JVP = jugular venous pressure
PCWP = pulmonary capillary wedge pressure
RAP = right atrial pressure

FIGURE 1 Assessing Hemodynamic Profiles in Patients With Heart Failure



(A) Hemodynamic profiles are based on volume status and adequacy of perfusion. **(B)** Potential therapeutic implications of classification of hemodynamic profiles. CI = cardiac index; PCWP = pulmonary capillary wedge pressure.

TABLE 1 Practical Tips When Assessing the JVP

1. Start by assessing the patient sitting upright to exclude a very high JVP, which may be more difficult to detect when at a lower angle.
2. Look for a waveform/pulsation rather than an actual venous structure. Shining a light tangentially across the neck may help you see the waveform.
3. If the patient is in bed, excess pillows may flex the neck, making the jugular veins hard to see; thus, we recommend leaving only 1 pillow. Slightly tipping the chin upward often improves the visibility of the jugular waveform.
4. Inspect both sides of the neck because the waveform may be seen better on either the right or the left. In a small minority of patients, the jugular venous waveform is seen best in the front of the neck (i.e., midline above the 2 clavicular heads).
5. If the internal jugular venous waveform is not visible, the external jugular vein can be used to estimate the JVP. Confirm a respirophasic component in the external jugular vein before accepting it as a measure of the JVP.
6. Assess the JVP with the patient at various angles off the horizontal (e.g., supine, at 30°–45°, sitting, or standing) until the pulsation is visible approximately halfway up the neck. Note that a high JVP can be hard to see in the supine position so a good practice is to also look at the neck veins with the patient sitting up.
7. To distinguish the carotid from the jugular venous impulse, apply pressure with your finger 1–2 inches below the impulse. If the pulsation disappears, it was the jugular vein; if the pulsation persists, it was the carotid artery.
8. A respirophasic pattern (typically a decrease with inspiration, but in some patients the JVP increases with inspiration, known as the Kussmaul sign) and positional changes (i.e., pulsation moves lower in the neck when the patient is more upright) also help establish the waveform as venous rather than arterial.
9. If the JVP does not seem elevated when the patient is supine, press on the abdomen to determine whether an HJR is present. Note that in many patients, the JVP will transiently rise (i.e., flicker upward for 1–2 s) when abdominal pressure is first applied but then return to normal. Although such a finding is not considered a positive HJR, which requires sustained elevation over 10 s, it will help the examiner be certain that the maximal height of the jugular venous column was seen (and the JVP was not underestimated).

HJR = hepatojugular reflux; JVP = jugular venous pressure.

TABLE 2 Utility of Clinical Findings in Detecting PCWP >22 mm Hg in Patients With Advanced Heart Failure in ESCAPE

Clinical Finding	Sensitivity	Specificity	PPV	NPV
Rales ≥1/3	15	89	69	38
Edema ≥2+	41	66	67	40
Orthopnea ≥2 pillows	86	25	66	51
JVP ≥12 mm Hg	65	64	75	52
HJR	83	27	65	49

Adapted with permission from Drazner et al. (7).

ESCAPE = Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness; NPV = negative predictive value; PCWP = pulmonary capillary wedge pressure; PPV = positive predictive value; other abbreviations as in Table 1.

patients with advanced HF (15). Thus, the JVP is useful not only in the assessment of fluid status but also in identifying patients with higher risk of adverse outcomes.

HEPATOJUGULAR REFLUX. The HJR is an increase in JVP by >3 cm sustained during 10 s of continuous pressure on the abdomen, with an abrupt fall after the pressure is released (16). Testing for HJR may improve detection of elevated ventricular filling pressures because the presence of HJR, in the absence of isolated right ventricular systolic dysfunction, reliably predicts PCWP >15 mm Hg (17). In a study of 52 patients referred for cardiac transplantation evaluation, 42% of patients had HJR, and all but 1 of these patients had PCWP ≥18 mm Hg. Additionally, there was good interobserver agreement in the assessment of HJR (8). In total, these findings suggest that HJR can be useful in identifying patients with elevated PCWP.

Similar to JVD, the presence of HJR indicates worse prognosis in patients with HF. In a post hoc analysis of ESCAPE, patients with persistent versus resolved HJR on discharge had a higher risk of 6-month mortality (univariate odds ratio: 2.2; 95% confidence interval: 1.2 to 3.9; p = 0.012). Furthermore, those with HJR and JVD on discharge had higher 6-month mortality compared with those with HJR alone (33.8% vs. 16.7%, respectively; p = 0.045) (18). Thus, evaluation for both JVD and HJR not only at admission but also at discharge provides prognostic information in patients with HF.

ORTHOPNEA. Orthopnea, or dyspnea when supine, was associated with elevated PCWP in ESCAPE, whether defined as ≥28 mm Hg, ≥30 mm Hg, or ≥32 mm Hg (7). Both orthopnea and JVD remained associated with PCWP ≥30 mm Hg independent of each other in multivariable models, suggesting they provide additive information. As such, during the clinical examination, we routinely assess our patients for orthopnea to estimate whether their left ventricular filling pressures are elevated.

A recent composite "orthodema" score based on the presence and severity of orthopnea and edema has been proposed (19). An increased orthodema score was associated with morbidity and mortality in patients with HF (19).

RESPONSE TO VALSALVA MANEUVER. The BP response to the Valsalva maneuver also allows estimation of the PCWP. In healthy individuals, the BP drops during the strain phase due to a decrease in pulmonary venous return to the left ventricle. In contrast, in patients with elevated left-sided filling pressure, a square-wave pattern of response is seen. The BP rises with strain and remains stably elevated throughout the strain, returning to baseline only when the strain is released. Note that there is no decrease in BP during the strain despite decreased pulmonary venous return, as occurs in healthy individuals, because the left ventricle remains adequately filled given the pre-strain elevated left

ventricular filling pressure (20,21). The square-wave response to the Valsalva maneuver was associated with the invasively measured PCWP (22–26). The square-wave response will not be present if there is isolated elevation in right ventricular filling pressure. Devices that allow quantitative assessment of BP in response to the Valsalva maneuver are being tested as a means of noninvasively determining left ventricular filling pressures (22,23,27).

BENDOPNEA. Bendopnea, as we recently defined in an advanced HF population (28), is a symptom of dyspnea that is elucidated by bending forward at the waist (**Figure 2**). Bendopnea was assessed by having the patient bend forward while sitting in a chair and touching one's feet with one's hands. Concurrently, an examiner told the patient not to hold his or her breath, then at 10-s intervals asked the patient if he or she was experiencing difficulty breathing. Bendopnea was considered present if dyspnea occurred within 30 s of bending (28). In the initial study of 102 patients with systolic HF undergoing right heart catheterization, bendopnea was present in a sizeable minority of patients (approximately one-third) and was associated with increased filling pressures, particularly in the setting of low CI (28). Four additional studies have confirmed that bendopnea is common (18% to 49%) in patients with HF, including those in a primary care clinic or HF clinic, those referred for cardiopulmonary exercise testing, and patients admitted with decompensated HF (29–32).

Bendopnea may have prognostic utility in HF. Patients with bendopnea had a higher minute ventilation to carbon dioxide production (VE/V_{CO_2}) slope (29), which is a validated prognostic marker in HF (33). In a study of 250 patients admitted with decompensated HF, bendopnea was associated with increased 6-month mortality in univariable but not multivariable analysis (30). In a study of ambulatory patients with HF, bendopnea was associated with an increased risk of a composite endpoint at one year of death, HF admission, inotrope initiation, left ventricular assist device implantation, or cardiac transplantation in univariable but not multivariable analysis (32). In that study, bendopnea was more strongly associated with short-term outcomes such as HF admission at 3 months.

Although bendopnea was associated with elevated filling pressures in patients with HF, it is not diagnostic of HF and may also occur in other disease processes. For example, bendopnea was reported with allergic bronchopulmonary aspergillosis (34) and likely can be present in patients with other pulmonary diseases or in the morbidly obese. These examples do not minimize the potential importance of bendopnea in assessing patients with HF, just as the presence of orthopnea and edema in obese subjects does not diminish the importance of these classic signs of HF.

FIGURE 2 Assessment of Bendopnea

A patient sits in a chair, bends at the waist, and touches his or her feet. Bendopnea is considered present if dyspnea occurs within 30 s of bending.

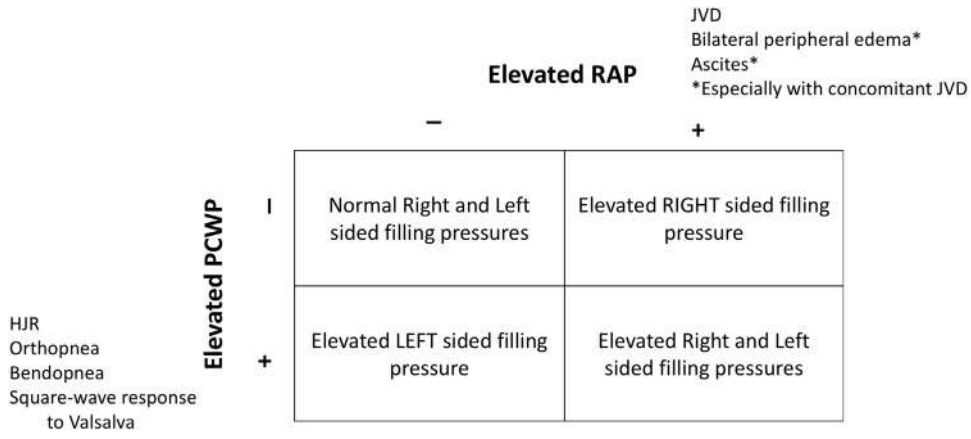
PERFUSION

In addition to assessing volume status, the clinical examination can be used to assess the adequacy of perfusion (**Figure 1A**). Although there are multiple useful clinical examination findings to determine an elevated PCWP (see "Volume status" section), there are fewer reliable findings to determine a low CI. Such findings include narrow pulse pressure, cool extremities, a global assessment of cold made by the clinician, and possibly bendopnea.

A low pulse pressure (systolic pressure - diastolic pressure) or a low proportional pulse pressure [(systolic pressure - diastolic pressure)/systolic pressure] can be a marker of a low CI. In a study of 50 patients with chronic HF undergoing hemodynamic assessment with right heart catheterization, the proportional pulse pressure correlated well with CI ($r = 0.82$; $p < 0.001$) (35). However, in ESCAPE, proportional pulse pressure <25% had good positive predictive values for low CI, but it was relatively infrequent and was not significantly associated with CI ≤2.2 l/min/m^2 (7).

There is even less supporting evidence for the other putative clinical findings of low CI. In ESCAPE, the clinician's overall assessment of a cold profile was significantly associated with CI ≤2.2 l/min/m^2 (odds ratio: 2.97; 95% confidence interval: 1.2 to 7.1; $p = 0.015$) (7). As described in the "bendopnea"

FIGURE 3 Classification of Congestion Based on Clinical Assessment of Elevated Right- or Left-Sided Filling Pressure



HJR = hepatojugular reflux; JVD = jugular venous distention; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure.

section, bendopnea was associated not only with elevated filling pressures but also with low CI (28). Cool extremities are consistent with poor perfusion, although it is important to recognize that an examiner's perception of the temperature of the patient's legs is dependent on whether the examiner's own hands are warm or cold. Furthermore, we have noted repeatedly that patients whose legs feel warm to the touch can have a low CI, suggesting that this finding has low sensitivity. Indeed, in ESCAPE, sensitivity was only 20% for this finding (7).

Overall, our clinical experience suggests that although ventricular filling pressures can be assessed with some confidence, estimation of the CI is more challenging. It is particularly important to recognize this limitation when assessing patients who are not responding favorably to therapy guided by the clinical examination. Specifically, a low CI should be considered in the differential diagnosis for worsening renal function during diuresis in a patient with decompensated HF, even if the clinical examination suggests the patient is well perfused.

PHYSICAL EXAMINATION FINDINGS LESS USEFUL IN ASSESSING HEMODYNAMICS IN HF

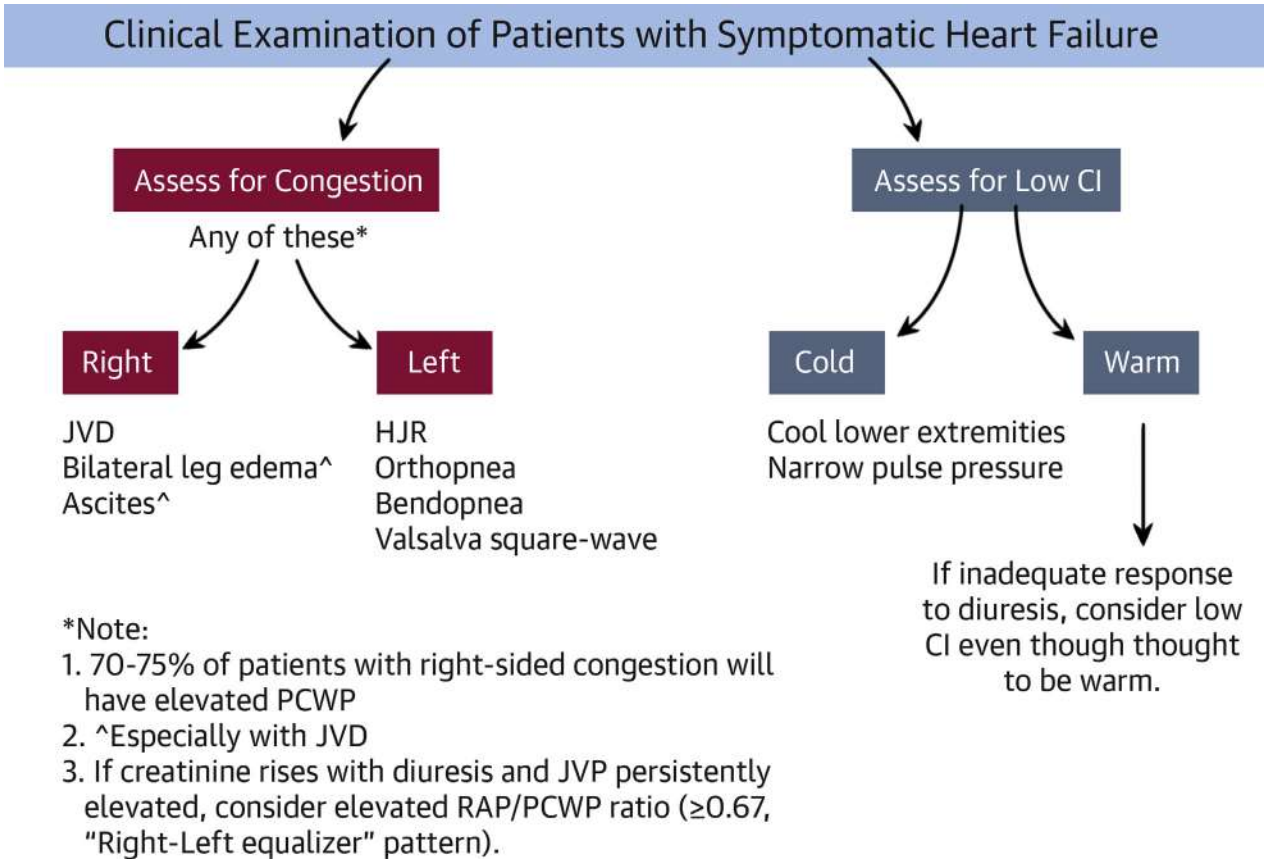
Although pulmonary rales, pleural effusions, and peripheral edema can be seen in decompensated HF, these findings have limitations. In advanced chronic HF, patients with elevated left ventricular filling pressures may not have rales (8,35-37) because of an increase in lymphatic drainage. Indeed, in our experience of caring for patients with advanced HF, outside of the setting of ischemia and "flash" pulmonary edema, rales are more often a reflection of a pulmonary process (e.g., pneumonia) than of pulmonary edema. Likewise, patients with HF may have clear lung fields on radiographic imaging despite having elevated filling pressures (36,38).

Peripheral or dependent edema may be common, but it represents extravascular rather than intravascular volume. Edema can be the result of another condition, such as venous insufficiency, obesity, lymphedema, nephrotic syndrome, or cirrhosis. Rapid accumulation of bilateral leg edema with associated weight gain in patients with a known history of HF usually does represent volume expansion, although it occurs late in the process of decompensation. Its specificity as a sign is increased in the presence of elevated JVP, but it should not be used alone to suggest elevated filling pressures (35).

UTILITY OF THE STEVENSON CLASSIFICATION

The Stevenson profiles provide prognostic information (1,7). In a single-center observational study, the admission H&P-assessed hemodynamic profile of B (warm and wet) or C (cold and wet) was an independent risk factor for the composite endpoint of mortality or urgent transplant at 1 year (1). In ESCAPE, physician assessment of discharge hemodynamic profiles of wet or cold, versus profile A (warm and dry), was associated with a 50% increased risk of rehospitalization or death, independent of other markers of disease severity (7). The value of serial assessment throughout hospitalization was evident, as the patients who were admitted wet or cold but were

CENTRAL ILLUSTRATION **Clinical Assessment of Hemodynamics in Patients With Heart Failure**



Thibodeau, J.T. et al. J Am Coll Cardiol HF. 2018;6(7):543-51.

This classification incorporates information on whether right- and/or left-sided ventricular filling pressures are estimated to be elevated. Note that both a disproportionately elevated ratio of RAP to PCWP (in the setting of persistently elevated JVD) and a low CI (despite a clinical assessment that the patient is warm) are in the differential diagnosis for worsening renal function during diuresis. CI = cardiac index; HJR = hepatojugular reflux; JVD = jugular venous distention; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure.

discharged in profile A did not have increased event rates after discharge, while the patients at high risk were those who were discharged while still cold or wet.

The Stevenson profile can also be used to guide therapy. Occasionally, a patient is admitted to the hospital with a presumed diagnosis of decompensated HF, yet the clinical examination suggests that the patient is compensated (profile A, warm and dry). In such cases, alternative causes of the patient's symptoms need to be considered (e.g., in the appropriate setting, amiodarone lung toxicity or pneumonia). The Stevenson profile can also be used to guide decision-making as to whether vasodilators or inotropes should be added to intravenous diuretics in patients admitted with decompensated HF. In our practice, if a patient is profile B, we administer diuretics, reasoning that there is no role for inotropic therapy when perfusion is adequate. In contrast, if the patient is profile C, we add either a vasodilator or inotrope to the diuretics (Figure 1B). Given the challenges in estimating CI (see "Perfusion" section), we recognize that some patients thought to be profile B are actually profile C, but we believe this framework is reasonable when choosing the initial therapeutic approach in hospitalized patients with decompensated HF.

INTEGRATING NATRIURETIC PEPTIDES WITH THE CLINICAL EXAMINATION

Even in the absence of clinical findings of congestion, patients with HF can have elevated left ventricular filling pressures, a condition called "hemodynamic

congestion" (2). One method for identifying hemodynamic congestion is to measure natriuretic peptides, neurohormones released in response to stretch of the left ventricular walls. Measurement of natriuretic peptides, in addition to the clinical examination, can improve the diagnosis and risk stratification of patients with HF (39). Although the hypothesis that adjusting therapy by serial measurement of natriuretic peptide levels rather than by clinical assessment could improve outcomes in patients with HF was attractive, this was not the case in the recently completed Guiding Evidence-Based Therapy Using Biomarker Intensified Treatment (GUIDE-IT) trial (40).

REFINING THE ASSESSMENT OF CONGESTION BASED ON WHETHER VENTRICULAR FILLING PRESSURES ARE ELEVATED ON THE RIGHT SIDE, LEFT SIDE, OR BOTH

Currently, most clinical markers of volume overload (e.g., JVD, peripheral edema, or ascites) reflect elevated right-sided filling pressures. Although it is true that an elevation in RAP mirrors an elevation in PCWP in most patients with HF, approximately 25% to 30% of patients have discordance between right- and left-sided filling pressures, with an isolated elevation on either side (41–44). Discordance can occur in those with preserved or reduced ejection fraction (41–44). The pattern of elevated filling pressures (right-sided only, left-sided only, or both) has some degree of consistency when reassessed over time (41,43).

To refine the clinical assessment of the congested state, patients can be classified based on whether the right- or left-sided ventricular filling pressures, or both, are elevated (**Figure 3**). Although this classification system may more accurately characterize the type of volume overload, whether patients with isolated right- or left-sided filling pressure elevation have a different response to treatment or prognosis than those with concordant pressures is not yet known. A disproportionate elevation of the right- to left-sided ventricular pressures, as assessed by an elevated RAP/PCWP ratio (termed a "right-left equalizer" pattern when ≥0.67), has been shown to be associated with impaired renal function (45) and worse outcome (43,46), providing preliminary support for this classification system. A disproportionately elevated ratio of RAP to PCWP should be in the differential diagnosis when a patient with persistently elevated JVP develops worsening renal function during diuresis. Further investigation of the characteristics and outcomes of these hemodynamic profiles is warranted. An overall approach to the clinical examination incorporating this approach is shown in the **Central Illustration**.

CONCLUSIONS

The clinical examination provides information regarding the hemodynamic state and prognosis of patients with HF. With regard to the patient's hemodynamic state, the clinical examination is more accurate in the assessment of elevated ventricular filling pressures than in the detection of a low CI. Currently, elevated left ventricular filling pressures are inferred largely from clinical findings related to an elevated RAP. An emerging classification system characterizes patients based on whether congestion occurs on the right side, left side, or both. Given these considerations, in patients with decompensated HF and worsening renal function during diuresis, a low CI (even if perceived to be well perfused) and a right-left equalizer pattern of ventricular filling pressures (when JVP is persistently elevated) should be included in the differential diagnosis. Further studies are needed to determine whether a classification based on right- versus left-sided congestion can be used to improve risk stratification and guide treatment decisions for patients with HF, and thereby improve outcomes.

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